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*Some Less Common Forms of Cerebrovascular Disease**

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Diseases involving cerebral blood vessels are very common. Arteriosclerosis and changes associated with hypertension are certainly the most common, and therefore the most important pathologic findings in these vessels. However, other diseases less frequently affect the cerebral blood vessels causing cerebral lesions. It is the purpose of this presentation to review a few of these less common cerebrovascular abnormalities.

Among the less common forms of cerebrovascular disease, the congenital or saccular aneurysm is probably the most frequent. An unusual complication of congenital aneurysm was observed in a patient with a ruptured saccular aneurysm on the anterior communicating artery and an unruptured aneurysm on the right internal carotid artery. This was a recent infarct in the inferomedial portion of the right temporal and occipital lobes, an area supplied by the posterior cerebral artery. As is commonly noted with any supratentorial mass, a herniation of the uncus beneath the free edge of the tentorium was present. It is suggested that in this patient the posterior cerebral artery was compressed between the herniated tissues and the free edge of the tentorium, producing the infarct.

In tuberculous meningitis the cerebral blood vessels are very commonly involved. Granulomatous inflammation, fibrinoid, thrombi, and subintimal fibrosis may be noted at various stages, primarily in arteries. These vascular changes not infrequently cause infarction of the brain and spinal cord. Such infarcts are more common than tuberculous encephalitis and are more likely to be the cause of clinical manifestations of parenchymal injury.

Infarcts of the brain due to a severe arteritis of the leptomeningeal vessels were observed in a patient with *Candida albicans* leptomeningitis. This was presumably related to long continued therapy with cortisone and a variety of antibiotics for an unrelated disease. The leptomeningeal infiltrate included polymorphonuclear neutrophilic leukocytes, many mesenchymal cells presumably of arachnoidal origin, and collagen and reticulum fibers. The arteries and veins were involved and often occluded by a similar inflammatory tissue. There was a superficial infiltration of the substance of the infarcted tissues by the leptomeningeal infiltrate. Hyphae were present in the leptomeninges, the affected vessels, and the infarcted tissues.

A rare vascular process with a tendency to involve the vessels of the brain to a major degree may be termed, at least in a descriptive sense, "granulomatous angiitis".

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These patients die as a result of cerebral infarction due to arterial occlusion by granulomatous tissue composed of proliferated endothelial cells, multinucleated giant cells of the foreign body and Langhans' type, and collagen and reticulum fibers. Fibrinoid and thrombi may be observed in these vessels. The veins are less involved. In other organs, similar changes may be noted in the vessels, and granulomas not specifically related to vessels are also seen. The nature of this process and its relationship to other diseases which it resembles to a greater or lesser degree are obscure.

Cerebrovascular lesions in lupus erythematosus may cause small infarcts. There is a marked proliferation of the endothelial cells of small arteries and arterioles, and the deposition of an amorphous eosinophilic or basophilic material resulting in occlusion of the lumen. The basophilic material is stained by the Feulgen method, and is presumably related to hematoxylin bodies.

Profound neurologic abnormalities are noted in Moschcowitz's disease (thrombotic thrombocytopenic purpura), and these may be related to the occlusion of capillaries, arterioles and venules by a homogeneous, granular, eosinophilic material. This is associated with a hyperplasia of endothelial cells. Rarely, a small hemorrhage is observed, but infarcts were not noted.

Similar profound neurologic changes were seen in a patient with a severe perivascular lymphocytic infiltration of a moderate number of cerebral vessels, and with a moderate lymphocytic infiltration of the walls of some of these vessels. This was associated with the presence of a large number of neoplastic cells in the lumen of these vessels. This patient had a malignant lymphoma of the reticulum cell type with ulceration of the tumor into the inferior vena cava. In the absence of any other pathologic change, it is suggested that the symptomatology was referable to the vascular inflammation described.

DISCUSSION

ABNER WOLF: The fascinating series of pathologic conditions involving the cerebral blood vessels that Dr. Feigin showed us is most illuminating.

He spoke first of the congenital aneurysms of the cerebral arteries, and I am sure he has wondered too, as we have, about the actual way in which they evolve. He illustrated the lack of medial development and of elastic tissue. It has also been suggested that perhaps anomalies of an accessory vessel, such as he doubtless has seen on the anterior communicating artery, might possibly be a source for such aneurysm formation. All of us have seen double anterior communicating arteries and it has been suggested that when such begin to develop perhaps one of the two arteries does so only abortively and its bud may be the source of the aneurysm. I should like to ask Dr. Feigin whether he has had some material which bears upon this hypothesis.

The common tuberculous arteritis which he illustrated leads only occasionally to infarction of the brain. With present day treatment of tuberculous meningitis we may expect to encounter permanent neurologic sequelae in some of the survivors of the disease, since some will surely have had infarcts of the brain such as Dr. Feigin has illustrated.

I wonder whether, in Dr. Feigin's case of thrombotic thrombocytopenia, there was some other disease process present which might have been a factor in damaging the walls of the small vessels, and precipitating the formation of the thrombi. It has been suggested that perhaps this is not a primary disease in the cerebral or other vessels, but rather a secondary syndrome. I remember that Dr. Spain reported seven cases from Columbia and found that six of the patients presented neurologic symptoms. Damage to the brain would seem to be frequent in this disease process.

IRWIN FEIGIN: I am grateful to Dr. Wolf for his comments. Abnormalities in the distribution of the cerebral arteries and abnormal accessory vessels are very common. I have not recognized the occurrence of an abortive stage which might subsequently lead to aneurysm formation. I have looked, with at least moderate care, for unruptured aneurysms in adults and in children. I may not have looked carefully enough to draw conclusions, but it does ap-

pear as though aneurysms are seen more often in adults than in children, and I believe that something must happen with advancing age which permits these aneurysms to develop. Perhaps the acquired changes can affect an abortive vessel as well as a vessel which shows the type of muscular and elastic defect that I tried to illustrate.

With respect to infarcts in tuberculous meningitis, my experience suggests that these do occur fairly frequently. However, my experience is limited, composed of treated cases for the most part, patients who have lived for extended periods with this infection.

In regard to the case of Moschcowitz's disease, I can only say I did not recognize any abnormality in the brain other than that demonstrated here. No other abnormality was recognized by the other members of the pathological staff who studied the other organs in this case. In short, we have recognized no other vascular change in our cases to which the changes of Moschcowitz's disease may be secondary.

VERA B. DOLGOPOL: I have seen a number of cases of tuberculous meningitis, both treated and at pre-treatment time, that showed extensive areas of infarction, sometimes caused by softening of the entire periventricular structures, resulting from obstruction of the vessels, even without any significant ventricular obstruction that could explain the degeneration of the tissues. I have seen cases that have been treated in which there was partial ventricular obstruction and which showed calcification of nerve cells as a part of the picture of infarction.

STANLEY J. KALLMAN: I am curious to know whether Dr. Feigin has noted any degree of medial dissection comparable to that seen in the larger arteries, and if not, why not?

MAX WACHSTEIN: My question concerns the 18 year old boy. I would like to know whether the lesion was limited to the brain, and if not, what was the underlying disease process?

ALFRED ANGRIST: I would like to ask a question about the infarction of the occipital lobe. Was there a thrombus in the posterior cerebral artery? In our group of cases we often failed to find the thrombus in this vessel, and the impression is that it is often a lesion of ischemia and hemorrhagic infarction on the basis of compression only. I do not know what Dr. Feigin's experience is, but I would be interested to know if he does not find that the point of actual narrowing of the vessel often corresponds to the site where the nerve compressed the vessel rather than as the result of the compression only of the herniated uncus itself.

This lesion of occipital infarction has not been stressed sufficiently, and I think it is rather common. I think Dr. Browder will verify this. We get it in trauma frequently. We have all seen it in all forms of extra- and intracerebral hemorrhages. It is sometimes bilateral. The old terminology of diffuse occipital contusion is of course a misnomer.

I think the case with the granulomatous lesion is a most amazing one, and I would be interested if Dr. Feigin would hazard a guess as to the mechanism involved.

SIGMUND L. WILENS: I would like to know if you have seen giant cell arteritis of the type that occurs in the temporal artery involving an intracranial vessel.

IRWIN FEIGIN: Dr. Kallman asked whether I recognized medial necrosis in congenital aneurysms. I have not, nor is it obvious to me why this must necessarily be present.

Dr. Angrist pointed out that uncal herniation is a common occurrence, which is certainly true, and frequently gives rise to hemorrhages in the brain stem. The posterior cerebral artery occlusion with uncal herniation is much less common, but is noted with increased pressure above the tentorium, such as may be seen with trauma, tumor, and in this case, a ruptured aneurysm. No thrombus was observed in the posterior cerebral artery in this case. I do not think that this proves the occlusion is due to compression, although I believe it was so caused, since it is often very difficult to find thrombi

in the vessels in the cerebral infarcts due to arteriosclerosis. Statistically, thrombi are found in a minority of cases.

Dr. Wilens has asked whether I have seen a case of giant cell arteritis involving the brain substance. I have not, but this has been reported by others. This question, and the questions by Dr. Angrist and Dr. Wachstein, relate to the case which we designated as granulomatous angiitis. The patient was an 18 year old boy who died in approximately three months as a direct result of this disease process. These two aspects, the age of the patient and the severity of the process, are unlike the situation we have been led to expect in temporal arteritis. There were lesions in the rest of the body in this case, but none of them produced any striking signs or symptoms clinically. The lesions involved to a major degree the intima of the pulmonary artery, and the aorta, as well as the coronary vessels. In addition, there were granulomatous lesions not directly related to blood vessels in the lungs, the lymph nodes, spleen, and liver, and some of these were rather bizarre. The lesion in the liver contained areas of necrosis. A great many sections were stained for tubercle bacilli, but none was found. The case is of interest in regard to its etiology and its relation to other more or less similar cases, as I tried to imply. Cases which resemble this one to a greater or lesser degree have been reported in the literature as sarcoidosis, granulomatous angiitis, and as allergic granulomatosis, the last designation being applied in cases associated with bronchial asthma. I must confess I do not fully understand this case or its relationship to the other cases mentioned.

ABNER WOLF: Dr. Wilens, involvement of the nervous system in giant cell arteritis was reported by Greenfield[1] some years ago.

REFERENCE

1. Greenfield, J. G. A case of giant cell arteritis, *Proc. Royal Soc. Med.* *44*:855-57, 1951.

Pathogenesis of Cerebral Lesions Resulting From Contact Trauma to the Head

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Contact trauma to the head may result in an instantaneous intracerebral lesion (e.g., hemorrhage), the pathophysiologic expressions of which are immediately apparent. The continuing presence of a compressive, hemorrhagic mass, in turn, may give rise to a series of tissue reactions attributable to the initial lesion but not directly consequent to the inciting blow. Such secondary and belated alterations can assume a far greater importance, clinically and pathologically, than the original traumatic hemorrhage.

Over a 24 year period, 41,549 patients who had sustained craniocerebral injury were admitted to the Kings County Hospital Neurosurgical Service. Of these cases, 822 were verified instances of subdural hemorrhages and are, because of their high mortality rate, of major concern in a study of head injury.

Autopsy examination in 29 cases of subdural hemorrhage indicates that a number of progressive intracerebral tissue changes developed which were not components of the primary head damage. It is these secondary lesions which help to explain the subtly advancing clinical decline and the late appearance of pertinent symptoms.

The extravasated mass of subdural blood can be considered as an extracerebral space-occupying tumor differing in essence from neoplasms or cysts only in the rapidity of development. The volume of hemorrhage appears to be one determinant of the ensuing clinical course; hematomata of less than 25 to 50 cc. are rarely associated with any appreciable neurologic disturbance. The presence of any larger extramedullary hemorrhage over the dorsal cerebral surfaces may profoundly affect the underlying hemispheres.

Such effects seem disproportionately greater than might be anticipated on the sole basis of the extracerebral blood volume. Ventricular air studies, for example, usually disclose a sizeable compression and deviation of the homolateral ventricle. Autopsies in these cases performed within a few days of precipitating injury show nothing to indicate any continuing expansion of the hematoma, but do reveal an independent feature responsible for the degree of ventricular distortion. The cerebral white matter ipsilateral to the hematoma is intensely swollen and more voluminous than the contralateral hemisphere. The edema beneath the hematoma cannot be attributed to any direct, circumscribed outgrowth of injury, since it is also seen in many cases of rapidly expanding subdural neoplasm.

The implicated white matter is visibly softened, pallid, but not grossly congested. Histologically, the deep transcerebral veins are dilated and the walls often infiltrated with inflammatory cells. The primacy of the transcerebral veins in the pathogenesis of the swelling of the cerebral white matter following development of extracerebral space-occupying masses is further suggested by postmortem vascular injection procedures. Preliminary studies in such cases have indicated that the functional patency of these vessels is diminished.

In traumatic cases with survival for a more prolonged period, the presumably reversible alterations in the white matter are now replaced by perivascular areas of tissue necrosis with abundant microglial participation. The individual foci are of microscopic dimension, but are present in great numbers. A collectively profound loss of myelin, principally perivenous, is encountered.

The rapid, unilateral increment in cerebral volume is directed mesially and inferiorly causing uncinate herniation around the free inner edge of the tentorium. The posterior cerebral artery courses within the apex of the triangle bounded by the lateral mesence-

phalic surface and the tentorium. As the vector of pressures is exerted through this triangle, the posterior cerebral artery is extrinsically compressed. In about 10 per cent of fatal cases of subdural hemorrhage, vascular insufficiency to the mesial occipital cortex supervenes with resultant encephalomalacia of the calcarine region.

Evaluation of the clinical course in cases of subdural hemorrhage not infrequently discloses a profound change abruptly developing hours or days after the primary hemorrhagic insult. The patient's state of consciousness is significantly depressed, the pupils assume a paralytic dilatation and an attitude of decerebrate rigidity is observed. These ominous clinical signs generally mirror secondary hemorrhages in the rostral brain stem. The hippocampal pressure cone responsible for compromise of the posterior cerebral artery is also directed toward the lateral mesencephalic surface, compressing the midbrain. The perpendicular vessels, arising from the upper basilar artery and mesencephalic (proximal posterior cerebral) arteries, and perforating the upper brain stem in the midsaggital ventral plane, appear to be the most susceptible to the adverse effects of this pressure. Foci of mural necrosis develop with resultant fusiform and spherical perivascular hemorrhages deep in the midbrain and pontine substance. The circumferential branches of the basilar artery supplying the lateral brain-stem tissues are not prone to this process, possibly because of the gradation in intraluminal pressures caused by the lack of any sharp transition between the main arterial trunk and the ultimate end-artery characteristic of the ventral midline system of vessels.

The vulnerability of the direct perforating vessels is further suggested by postmortem radiopaque injection studies. Artificially induced hypertension within the basilar artery often leads to perivascular extravasations of the injected medium about the midline arterial arborization but generally not about the vessels which first pursue a lateral and dorsal pathway before entering the brain stem.

There are numerous indications that hemorrhages of the midbrain and pons are not the direct concomitant of head trauma but rather a sequel to the inciting subdural space-occupying mass. The infratentorial hemorrhages are invariably more recent than the subdural hemorrhages, as determined histologically. Further, such brain stem hemorrhages follow the development of numerous non-traumatic supratentorial, rapidly expanding masses such as spontaneous cerebral hemorrhages and neoplasms.

On rare occasions patients with subdural hematoma have survived the critical and disruptive brain stem hemorrhages. These individuals generally remain in a vegetative coma, appear chronically decerebrate, and usually succumb from some intercurrent infection. Autopsy examination discloses no residual cerebral swelling but extensive ipsilateral atrophy of white matter with coalescent zones of demyelination and tissue destruction. The midbrain and pontine tegmentum contain cavitary regions of old hemorrhagic encephalomalacia.

It is conceivable that the untreated phase of cerebral swelling can subside without incurring secondary brain stem diapedeses. The diffuse cerebral white matter damage, however, persists and a psychiatric disorder of the patient results. Numerous institutions for the care of the mentally ill have reported an unusually high autopsy incidence of chronic subdural hematomata in patients not suspected of having sustained any previous traumatic lesion.

The critical importance of the cerebral swelling phase in traumatic cases is emphasized by the frequent observation that surgical evacuation of the subdural collection of blood may not reverse the deteriorating course of events. Thus, brain stem hemorrhages sometimes develop even after removal of the original hematoma. The progressive pathologic changes, once begun, may not be diverted by removal of the initiating factor, suggesting that the original subdural hemorrhage assumes a position of secondary importance once the vicious cycle of cerebral swelling commences.

Summary

The clinical course of subdural hemorrhage, unattended by surgical intervention, is generally a variable period of lucidity,

gradual suppression of the conscious state, deepening coma, decerebration, and death. It would be difficult to confer the full responsibility for this upon a subdural hemorhagic mass which is often less than 100 cc. in volume. The idea of posthemorrhagic expansion of the subdural hematoma is not consistent with autopsy observations, yet the quality of post-traumatic progression is undeniable. It is probable that the subdural compressive mass initiates a chain of irreversible events within the subjacent cerebral hemisphere. The deep cerebral veins are altered and venous stasis ensues. Perivenous edema, demyelination and necrosis result and the ipsilateral hemisphere becomes more voluminous. It is suggested that this intracerebral expansion rather than the preliminary subdural hemorrhage instigates the deterioration noted clinically, and inaugurates what is often an irreversible progression within the rigid cranial cavity. The delayed expansion of the cerebral hemisphere encroaches upon the posterior cerebral artery causing calcarine malacia and upon the mesencephalon inducing terminal hemorrhages in the upper brain stem. Other traumatic and non-traumatic primary lesions may also begin the same process and terminate in fatal brain-stem bleeding. The intermediate but fundamental link of cerebral swelling is, however, the necessary substrate and the determining factor in the ultimate outcome in each case.

DISCUSSION

E. JEFFERSON BROWDER: It certainly would be much more appropriate for a pathologist to open this discussion, particularly someone who is not so completely in accord with what Dr. Aronson has just said. For some 25 years we have been looking at cerebral swelling which is found in almost every instance of major subdural collection with fatal outcome, and it now seems that Drs. Aronson and Kaplan are beginning to unearth the pathogenesis of this edema. As you recall, in the early thirties it was postulated by Gardner[1] that the subdural membrane that forms about such collections is a semi-permeable one. The enclosed blood breaks down; there is created a high osmotic state within the subdural collection; cerebrospinal fluid, or regional fluid at least, is drawn, and there ensues at a later date, as is recognized in this syndrome, progressive stupor, coma and death, unless the subdural mass is drained off. Subsequently, there emanated, from the Boston City Hospital in particular, studies on subdural fluids that seemed to give considerable support to Gardner's concept. Over the years it has almost been accepted as a truism that the patients who have a clinical course of trauma, immediate loss of consciousness, subsequent lightening of the stupor, then, at a later date, stupor, coma, and if untreated, death, have followed a course consistent with this postulate of Gardner's. Dr. Aronson has pointed out the gross swelling of the brain underlying subdural collections. Dr. Kaplan has previously demonstrated an elaborate transcerebral venous system, connecting the veins of the cortex with those of the internal venous system of the brain. It would seem that the surface or subdural collection compresses the brain, thereby impairing its circulation, especially the venous, with consequent swelling of the white matter directly beneath the compressed cortex. It is quite logical to attribute the latent symptomatology of patients with subdural collections to the intracerebral edema as demonstrated by Dr. Aronson.

ABRAHAM M. RABINER: As Dr. Browder has indicated, we have been concerned with this problem of the clinical symptomatology of subdural hematoma. The mechanism and basis are rather unusual and perplexing. There are features of the clinical picture that are most important, especially in the later years of life. This we have recently stressed.[2] In so-called cerebral vascular disease, there is usually an acute onset with or without coma. Coma, when present, clears up within a matter of minutes or a few hours. If coma persists for more than 24 hours, the prognosis is unfavorable for return to a conscious state.

In the great majority of cases following the acute onset, the patient, fully conscious, has a residual motor or sensory deficit, or both, usually hemiplegia, which either remains stationary or improves. Not infre-

quently, and particularly in the later years of life, such patients who have had an acute onset, with or without unconsciousness, do well for a time, but then go into a drowsy state from which they may emerge and be clear for a while, only to again become somnolent. The physician treating such patients knows that there is little if any likelihood of recovery. This clinical picture has been labelled cerebral arteriosclerosis.

Even in the presence of definite cardiovascular renal disease, patients with such a clinical sequence of events should be afforded the benefit of burr-hole investigation by a neurosurgeon. A chronic subdural hematoma can be evacuated and a patient previously consigned to certain death, recovers. Even at age 80, salvaging a life is worth while.

ALFRED ANGRIST: This has been a beautiful presentation. I would like to prod Dr. Aronson into venturing more than just a pathologic demonstration, by asking for an expression of opinion as to the mechanism of the edema. Does he think it happens as a result of the initial trauma, this causing the changes he demonstrates immediately, or are they later secondary pathologic lesions following upon initial cerebral injury? Does the edema, and the other morphologic changes, occur as the result of lack of drainage through the torn cerebral veins and the impaired venous drainage by compression by the subdural hemorrhage? Did you see this kind of picture in patients who die very early after trauma with a subdural hemorrhage? In my own expericene, it is best seen in those who survive for a while. Then, have you any correlation as to the nature and the degree of trauma and the presence and the degree of the edema and the other pathologic changes? I think that would be very pertinent information. For instance, is there any correlation between those who had fractured skulls and this intracerebral lesion,—using skull fracture as a mere indication of the degree of trauma? Did you find this lesion in those cases where, from the history, the trauma was relatively minimal? Then the physical distortion is more that of the rotation type that tears a bridging vein in contrast to a direct severe traumatic episode that involves the cerebrum underneath.

Concerning the pontine lesions, we have something to complicate the situation, i.e., early immediate and later hemorrhages. We find one form of pontine hemorrhages with an uncinate herniation. Did you find more arterial than venous foci of bleeding in the cases that died only after time for the herniation of the uncinate gyrus and the edema causing it to develop? The lesion in the pons is not due to the initial trauma, but depends upon the secondary edema and the uncinate gyrus herniation, and the compression of venous return that also follows. Another type of pontine hemorrhages, the immediate pontine "blow-outs", I think are caused by the alternating positive and negative waves of pressure that occur at the time of the trauma, with a resulting momentary arrest or reversal of the flow of blood with rupture of the vessels. Both the early and late pontine hemorrhages are then due to imbalance between the pressures in the cerebrospinal system, the arteries and the veins.

This physiologic approach to the subject by Drs. Aronson and Kaplan may direct our therapy more intelligently. If the edema is so important, as it definitely is,—and incidentally I think the edema also involves the opposite hemisphere not uncommonly,—if the edema is important, I wonder if the "old-fashioned decompression" with a flap would help some of the cases.

I do want to say that we are all indebted to the authors for this presentation.

STANLEY M. ARONSON: Before answering a few of Dr. Angrist's questions, I would like to make some preliminary comments. I have avoided the use of the word "edema", and have used a somewhat safer word, namely, swelling, which is more accurately descriptive without implying the actual pathogenesis of the lesion or its physiologic nature. I do not think edema *per se* describes the process. While there may conceivably be increased fluid content reflected in an increase in the hemispheric volume, there are a number of defined and occasionally reversible tissue changes which obviously go beyond the realm of mere edema.

Thus, we have used the word swelling as a more inclusive term.

In the last two years we have had but 29 autopsies of significant subdural hemorrhage, and therefore a direct correlation between the extent and degree of damage and the volume of hemorrhage I think would be speculative until we have further cases. We have noted, however, (and some of the pictures I have used this evening are derived from such cases), that reasonably insignificant trauma has been associated with fairly massive hemorrhage. I believe there is very little correlation between the magnitude of trauma and the resulting degree of hemorrhagic volume.

ALFRED ANGRIST: I am referring to changes in the cerebrum itself.

STANLEY M. ARONSON: Again I would be reluctant to make correlations related to the extent of trauma. The patients enter in a stuporous state and an accurate history is difficult to obtain.

As to the mechanism of the swelling, we do not believe it is secondary to the immediate trauma. We feel, without sufficient evidence as yet, that the presence of a mass has an effect on the subjacent deep transcerebral veins. We think this because we have seen some instances of rapidly expanding dural neoplasms with comparable changes in the white matter and therefore cannot ascribe such changes to trauma.

You asked if specimens seen immediately after trauma showed swelling. We have seen swelling in autopsies as early as four to six hours after the hemorrhagic insult; in some instances of epidural hemorrhage, very distinct morphologic cerebral changes were evident six hours after the automobile accident precipitating the injury. Concerning the relationship of the degree of trauma to the degree of edema, we have no way of knowing because of insufficient cases.

You mentioned the possibility of some correlation with skull fracture. We have noted that the mortality rate increases when there is an associated skull fracture. If the fracture involves the temporal or occipital bones there is no great increase in the mortality; however, when it involves the basilar parts of the skull the mortality rate rises significantly.

Your question about pontine lesions: are they arterial or venous? The distribution would indicate to us that they are arterial, realizing that we are going in the face of accepted classical teaching; most textbooks state it is venous, but we have to accept our histologic observations when we can demonstrate an artery in the midst of a fusiform brain stem hemorrhage.

The time interval required for a secondary brain stem hemorrhage to emerge in relation to the degree of cerebral swelling is rather difficult to determine because the abrupt change in the clinical course is not too evident from the records, and the precise hour when the patient shows alteration in his state of consciousness is often obscure. We have seen, however, one case in which a patient died within 14 hours after trauma with cerebral swelling and brain stem hemorrhage already present, so we presume the entire process can occur within 14 hours. We have seen secondary brain stem diapedesis develop following massive cerebral hemorrhage of hypertensive etiology within six hours after the apoplectic crisis.

Your last question concerned decompression. I think it would be compounding an error to suggest decompression, for reasons which I think Dr. Browder would be more competent to explain.

E. JEFFERSON BROWDER: I suppose if one could turn down a flap comprising half the skull, in some instances it would be of some advantage, but certainly to make a classical subtemporal opening which only permits a small segment or portion of the brain to herniate would, as Dr. Aronson said, compound a felony and only add more insult to the situation, with more venous stasis, thrombosis, and I presume more of this so-called cerebral edema we have been talking about.

REFERENCES

1. Gardner, W. J. Traumatic subdural hematoma with particular reference to the latent interval, *Arch. Neurol. Psychiat. 27*:847-58, 1932.
2. Rabiner, A. M. and Schachter, I. B. Chronic subdural hematoma in guise of cerebral arteriosclerosis, *N. Y. State J. Med. 56*:2222-24, 1956.